A Report on Toxocara Canis Werner, 1782[1]

by Gloria A. Webster[2]

VETERINARIANS frequently encounter *Toxocara canis,* the intestinal ascarid worm in dogs, for this helminth is extremely common in young pups. As *Toxocara* is readily removed by the administration of anthelmintics and is rarely a serious problem in animals over six months of age, the general consensus has been that the parasite is innocuous; it therefore received only the cursory attention of the veterinary profession. When it was determined (1) (2) that prenatal infections of *T. canis* were responsible for a large percentage of still births and early mortalities in dog and fox pups, a more serious attitude was adopted. In addition, it has recently been found that *Toxocara* is one of the etiological agents of the clinical syndrome in children temed visceral larva migrams (3). The recognition of *T. canis* as an economic and public health problem has led to extensive researches on its life history and biology which had not previously been thoroughly investigated.

Toxocara is a cosmopolitan parasite widely distributed throughout the semitropic and temperate zones. The incidence of infection fluctuates with seasonal variations (4), but in general, *Toxocara* tends to be more prevalent in warm climates than in extreme northern areas; in the Northwest Territories *Toxocara* is rarely found. *T. canis* is primarily restricted to the family Canidae and reports of its occurrence in Felidae must be regarded with some scepticism, for considerable taxonomic confusion existed prior to the separation of *T. canis* and *T. cati* as distinct species.

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LIFE HISTORY

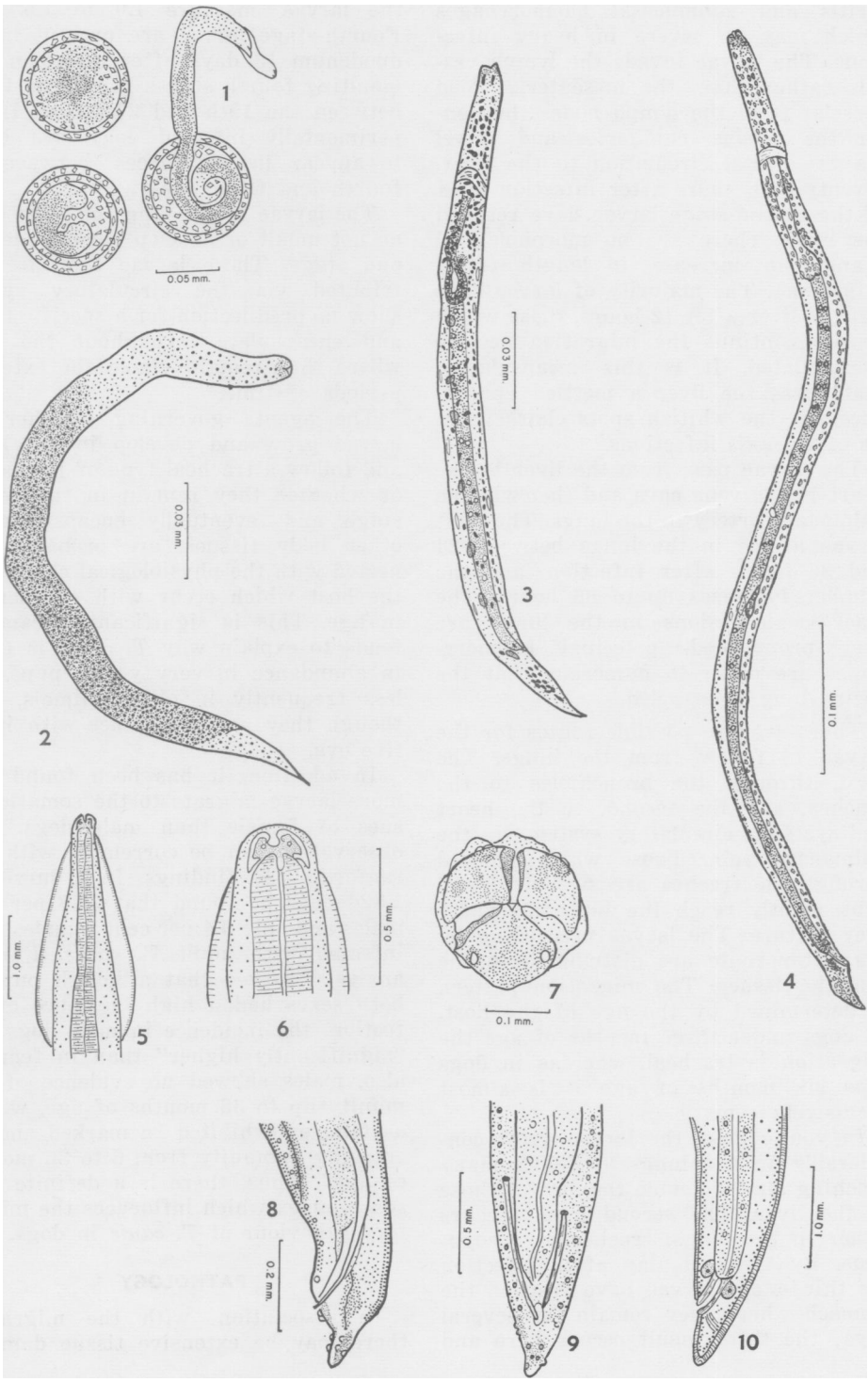
Toxocara is a member of the family Ascarididae and therefore a relative of *Ascaris lumbricoides,* the round worm in pigs. The life history of *Ascaris* is well known, and its liver-lung-trachea-oesophagus migration has become a classical example of larval migration. Like *Ascaris, Toxocara* also migrates through the tissues of its host, but the pattern is more complex.

The ova of *T. canis* average 0.087 mm. in length by 0.075 mm. in width; the shape varies from spherical to ovoid. The surface contour of the egg appears corrugated due to the mammilation of the protein layer of the shell, and because of this the eggs are opaque. Development of the eggs takes place outside of the host. The rate at which development proceeds depends upon the environmental conditions; the eggs are very resistant to low temperatures (5), and embryonation may be either temporarily halted or retarded. Moisture is essential for development, direct exposure to sunlight or dessication cause disintegration. Under optimum conditions embryos are present after six days and the first moult occurs within the egg on about the 12th day. The eggs are not infective until this moult has taken place.

When ova containing second-stage larvae are ingested by a dog the larvae hatch out in the small intestine. The freed larvae penetrate the intestinal wall, and in so doing produce an en-

PLATE I

Fig. 1. Eggs of Toxocara canis. **Fig. 2. First-stage larva of** Toxocara canis. **Fig. 3. Second-stage larva of** Toxocara canis. **Fig. 4. Third-stage larva of** Toxocara canis. **Fig. 5. Anterior end of adult** T. canis **showing lateral alae. Fig. 6. Dorsal view of anterior end of adult** T. canis. **Fig. 7. En face view of adult** T. canis. **Fig. 8. Male tail of adult** T. canis. **Lateral view. Fig. 9. Male tail of adult** T. canis. **Ventral view. Fig. 10. Posterior end of adult female** T. canis. **Lateral view.**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teritis and submucosal haemorrhages which may be severe in heavy infections. The larvae invade the lymph vessels rather than the mesenteric blood vessels; from the lymph nodes they enter the venous capillaries and travel via the portal circulation to the liver. Twenty-four hours after infection most of the second-stage larvae have reached the liver. There are no morphological changes or increase in length (0.41-0.49 mm.). The majority of larvae have left the liver after 12 hours, those which do not continue the migration become encapsulated. It is this encapsulation that gives the liver a mottled appearance and the whitish spots characteristic of *T. canis* infections.

The larvae pass from the liver to the heart in the vena cava and then via the pulmonary artery to the lungs. The first larvae arrive in the lungs between 24 and 36 hours after infection, and the number increases up to 96 hours. The macroscopic lesions in the lungs are very pronounced, petechial haemorrhages are often so numerous that the entire lung is stippled.

There are two possible routes for the larvae to follow from the lungs. The first, through the bronchioles to the trachea, and the second, to the heart and systemic circulatory system via the pulmonary vein. Those which proceed through the trachea are swallowed and subsequently reach the intestine where they mature. The larvae proceeding by the latter route are distributed to the somatic tissues. The migration pattern is determined by the age of the host. In dogs under three months of age the migration is tracheal, whereas in dogs over six months of age it is almost exclusively somatic.

In young dogs the larvae grow considerably in the lungs (0.8-0.95 mm.) reaching almost double the size of those in the liver. The second moult occurs either in the lungs, trachea or oesophagus. By the 10th day after infection the third-stage larvae have reached the stomach where they remain for several days; the third moult occurs here and the larvae measure 1.0 to 1.5 mm. Fourth-stage larvae are present in the duodenum 13 days after infection, and moulting fourth stages have been found between the 19th and 27th day. In experimentally infected dogs, ova begin to appear in the faeces between the fourth and fifth week.

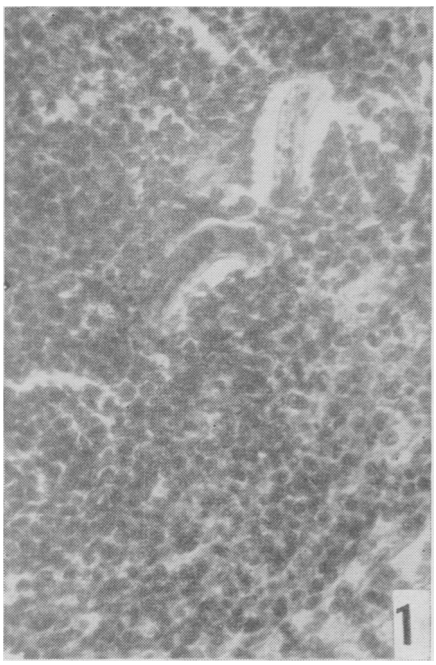
The larvae in the lungs of older dogs, do not moult or develop beyond the second stage. These larvae that are distributed via the circulatory system show no predilection for a specific tissue and encapsulate throughout the host, where they remain viable for extended periods of time.

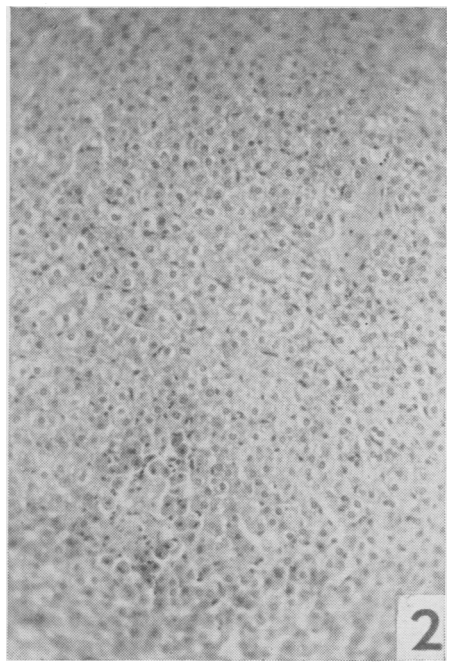
The agents governing whether the larvae grow and develop in the lungs and follow a tracheal type of migration, or whether they remain in the second stage and eventually encapsulate in other body tissues, are probably connected with the physiological changes in the host which occur with an increase in age. This is significant, because it tends to explain why *T. canis* is found in abundance in very young pups, and less frequently in older animals, even though they are in contact with infective ova.

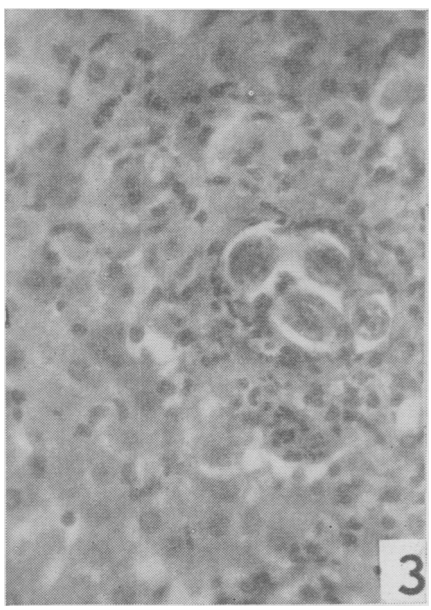
In addition, it has been found that more larvae migrate to the somatic tissues of female than male dogs. This observation can be correlated with Ehrenford's (4) findings. In a survey of 1,324 dogs he found that 32.8 per cent males and only 9.4 per cent females were infected with adult *T. canis*. Further analysis showed that although pups of both sexes had a high incidence of infection, the incidence in male dogs was "significantly higher" than in females, also, males showed no evidence of immunity up to 36 months of age, whereas females exhibited "a marked and increasing immunity from 6 to 36 months of age". Thus, there is a definite age-sex complex which influences the migratory behaviour of *T. canis* in dogs.

PATHOLOGY

In association with the migration there may be extensive tissue damage,







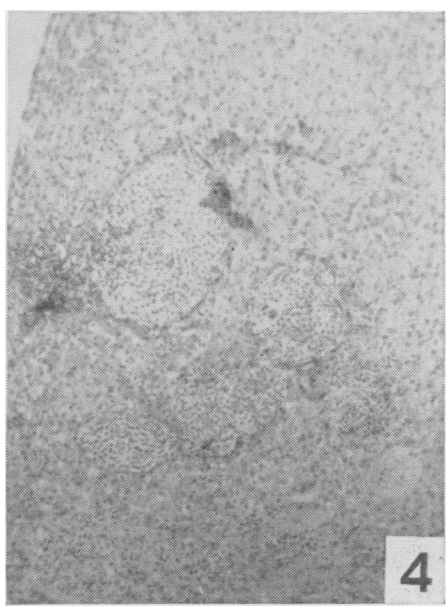


PLATE II

Fig. 1. Second-stage larva of Toxocara canis in liver surrounded by dense nodule of leucocytes (x480).

Fig. 2. Liver section showing haemorrhage, fatty degeneration of tissue and cell necrosis. (x120).

Fig. 3 Encapsulated T. canis larva in liver (x120)

Fig. 4. Liver section showing tissue repair by proliferation of connective tissue. (x120).

and the histopathology of the liver and lungs has been recorded by several investigators (6) (7).

Seventy-two hours after infection, liver sections show free larvae in the parenchyma and an infiltration of leucocytes. The number of leucocytes continues to increase during the next 24 to 48 hours, the predominant cells being polymorphonuclear leucocytes, monocytes and eosinophiles. There is a tendency for these cells to accumulate into nodules, and although occasionally a larva can be seen in their midst, more often they are free in the tissue. The tissue becomes necrotic, haemorrhages are common, and there is a marked fatty degeneration of the liver cells. The first vestiges of a capsule are seen about the 10th day, when the larva becomes surrounded by a thin fibrous capsule. Coinciding with the capsule formation, is the withdrawal of some of the leucocytes from the area. The regenerative process of the liver tissue begins about three weeks after infection and foci of liver cell degeneration are repaired by fibrous tissue proliferation.

There is also an infiltration of leucocytes in the lungs, with a high percentage of eosinophiles. These cells congregate, forming dense nodules. In light infections there is a lobular pneumonia and vascular congestion; in heavy infections the pneumonia becomes severe and there is an exudate containing red blood cells, epithelial cells, mucus and larvae in the bronchioles and alveoli. Haemorrhagic areas are common and some sections show complete tissue degeneration.

Unless there are large numbers of adult worms in the lumen of the intestine the pathology is usually limited to a moderate enteritis. When large masses of worms are present, they may produce an intestinal obstruction which can result in the rupture of the intestine.

PRENATAL INFECTION

Prenatal infection is important in

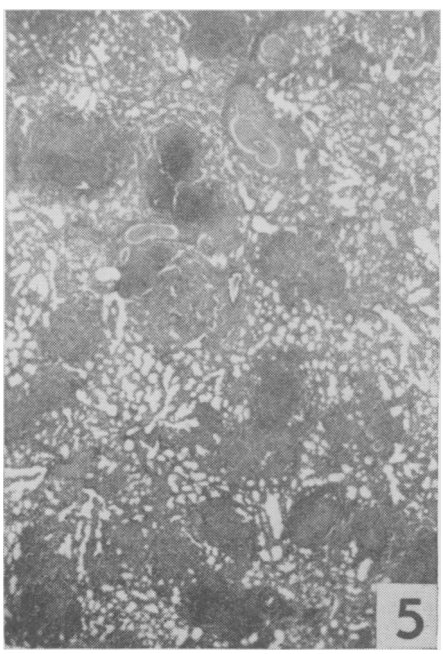


Fig. 5. Section of lung with numerous nodules resulting from leucocytic infiltration after invasion of tissue by T. canis (x15).

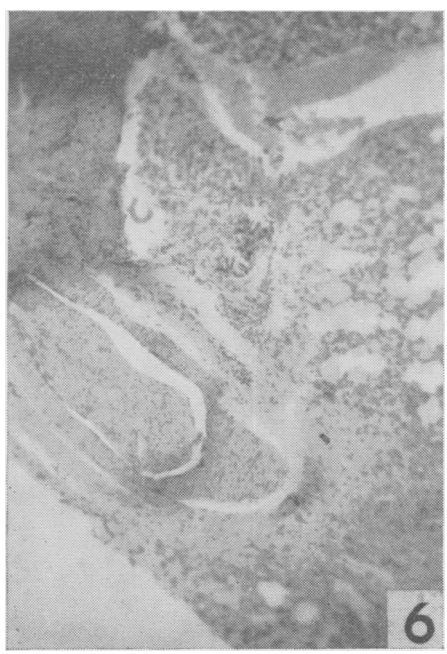


Fig. 6. Section of congested lung tissue with a single T. canis larva.

the epizoology of *T. canis*. Petrov (2) found an extremely high incidence of *T. canis* in newborn and young silver foxes; the infection decreased with increasing age, until it disappeared when the animals were four years old. In studying the incidence in stillborn and living pups, Nifontov (1) reported that intrauterine infection could be responsible for infection rates as high as 89.7 per cent in breeding establishments.

Intrauterine infection can be established either by the ingestion of infective ova during the gestation period or, through the reactivation of the second-stage larvae encapsulated in the somatic tissues. In the first case, the larvae during their migration through the host are carried to the placenta via the circulatory system; once they have reached the maternal capillaries of the placenta they penetrate the thin layer of tissue separating the maternal and foetal blood. The zonary deciduate type of placenta in carnivores is well adapted for the transference of larvae from maternal to foetal tissue.

Courmelles (8) observed that female dogs clinically free from adult *Toxocara* produced infected litters, and he considered that there was a relationship between the life history of the parasite and prenatal infection. Yutuc (9) found that bitches, free from intestinal infection and relatively isolated from any contact with helminth ova during gestation, still produced litters with *T. canis* and *Ancylostoma caninum*. This led him to postulate that: "... the infection of the bitches which contributed to the parasitism of the pups had been acquired prior to the initiation of the gestation period, a *pregestation infection*". He considered that the larvae, immobilized in the somatic tissues of the host became activated during pregnancy due to the lowered resistance of the host and the "debilitating effects of pregnancy", and travelled via the maternal circulatory system to the foetal tissue. Yutuc's hypothesis that larvae in the somatic tissues serve as a source of intrauterine infection has just recently been confirmed by the writer (10).

The age-sex migration complex is directly connected with this type of intrauterine infection. That more larvae migrate to the somatic tissues of female than male dogs and that, when the dogs are beyond six months of age, there is a marked tendency towards a somatic rather than a tracheal migration are important facts in that they ensure the maintenance of a high level of larvae in the tissues; the habits of dogs make frequent contact with *T. canis* eggs unavoidable. The mechanism by which encapsulated *T. canis* larvae are released from the somatic tissues is unknown, but it is thought that it may be connected with the hormone changes which accompany pregnancy.

The route taken by the larvae once they have entered the foetus is not known; however, the second-stage larvae do not migrate beyond the liver and lungs, intestinal infection is established four to six days after parturition (11) (12), (13).

VISCERAL LARVA MIGRANS

Within the past decade, considerable attention has been focused on host-foreign parasites, that is, those parasites occurring in a host other than their normal one. A parasite that has established a satisfactory host-parasite relationship, that is, has become well adapted to its environment as a result of a long association between host and parasite, usually does not cause extensive damage to its host. Organisms, however, which have been introduced into a host fairly recently in evolutionary time or which gain entrance to an abnormal host, tend to produce harmful effects due to their erratic behaviour, and this applies particularly to their aberrent migration. Beaver et al (3) have adopted the term visceral larva migrans to denote a prolonged internal migration of a larval parasite in an abnormal host.

When infective ova of *Toxocara* are ingested by an abnormal host, the larvae

migrate through the liver and lungs and are eventually distributed to the somatic tissue. Man is no exception, and Smith and Beaver (14) have definitely established that *T. canis* is one of the causative agents of human visceral larva migrans. This disease is found primarily in young children and can be correlated with their tendency to ingest quantities of soil, which frequently is contaminated with *Toxocara* ova.

The clinical symptoms, according to Beaver (15) ". . . are variable with the exception of eosinophilia which is always persistent and well above normal limits; levels above 50 per cent are common. More often medical attention has been sought because of intermittent fever, cough, muscle and joint pains, abdominal pain or neurological disturbances. Hepatomegaly and pneumonitis generally have been noted in the earlier stages of the infection."

As *T. canis* seldom becomes established in the human intestine there is no simple technique for direct diagnosis, the only reliable method today is the location and identification (16) (17) of larvae in sections taken from liver biopsy. Satisfactory serological techniques have not yet been developed.

Recently, there has been some discussion concerning the possibility of transmission of micro-organisms by helminths, and it has been determined that nematodes may carry viruses from one host to another (16) (18) (19). It is also thought that there may be a possible relationship between a posterior paralysis in pigs and the incidence of *Ascaris suum*, and one case of poliomyelitis has been reported in conjunction with an ascaris-caused visceral larva migrans (18). The present status of knowledge makes any conclusions impossible, but the fact that nematode larvae invade such a wide variety of tissues makes it probable that if they are not actually involved in the transmission of viruses, they certainly can facilitate their invasion.

As *Toxocara* has a wide geographical distribution visceral larva migrans is a potential problem wherever dogs are commonly infected. To date, cases have been reported from various sections of the United States, Great Britain, and Puerto Rico; recent enquiries from medical and veterinary personnel in western Canada indicate its probable occurrence in this country.

At present there are no adequate control measures, the resistant nature of the egg-shell makes spraying with chemicals impractical. Adult education in hygiene although effective, is difficult. This leaves the following suggestion as perhaps the best that can be offered. A system of mandatory worming imposed on dog breeders and kennel owners before releasing dogs would substantially decrease the potential number of eggs. Likewise, if veterinarians were cognizant of the threat to human health, routine faecal examination of animals taken into their clinics and subsequent treatment given in cases requiring such, would also reduce the possibility of human infection and the worm burdens in the canine population. While this method would be far from 100 per cent perfect, it seems the most logical and practical course open to us at this time.

REFERENCES

1. NIFONTOV, S., (Importance of intra-uterine invasion in the epizoology of **Toxocara** infection in dogs.) In Russian. Veterinariia, 1949, 26, 32-34.

2. PETROV, A., (Importance de l'invasion intruterine dans l'epizootologie de la toxocarose des renards noirs-argentes.) In Russian. Vestnik Sel'skokhoz. Nauk. Veterinariia, 1941, (3), 84-92.

3. BEAVER, P., SNYDER, H., CARRERA, G., DENT, J. and LAFFERTY, J. Chronic eosinophylia due to visceral larva migrans. Report of three cases. Pediatrics, 1952, 9, 7-19.

4. EHRENFORD, F. Canine ascariasis — a potential zoonosis. J. Parasitol. (Suppl.) 1956, 42, p. 12.

5. OWEN, W., Factors that influence the development and survival of the ova of an ascarid roundworm, **Toxocara canis** (Werner, 1782) Stiles, 1905, under field conditions. Tech. Bull. (71) Minnesota Agric. Exper. Stat., 1930, 25 pp.

6. HOEPPLI, R. Die durch Ascarislarven bei experimenteller infektion im Tierkorper bewirkten anatomischen Veranderungen. Virchow's Arch. Path. Anat., 1923, 244, 159-182.

7. HOEPPLI, R., FENG, L. and LI, F., Histological reactions in the liver of mice due to larvae of different **Ascaris** species. Peking Natural Hist. Bull., 1949, 18, 119-132.

8. COURMELLES, F. Comment pénètrent les **Ascaris** chez les tres jeunes chiens? Rev. Pathol. Comparee et Hyg. Gen., 1935, 35, 1332-1336.

9. YUTUC, L., Prenatal infection of dogs with Ascarids, **Toxocara canis** and hookworms, **Ancylostoma caninum.** J. Parasitol., 1949, 35, 358-360.

10. WEBSTER, G., On prenatal infection and the migration of **Toxocara canis** Werner, 1782, in dogs. Can. J. Zool. 1958, 36, 435-440.

11. NODA, R., On prenatal infection of dogs with ascarids, **Toxocara canis.** Bull. Naniwa Univ., 1954, Ser. B, 4, 111-119.

12. SHILLINGER, J. and CRAM, E., Parasitic infestation of dogs before birth. J. Amer. Vet. Med. Assoc., N.S.V., 1922, 16, 63, 200-203.

13. YOKOGAWA, S., On ascariasis and the life-history of **Ascaris.** (In Japanese. Taiwan Igekkwai Zasshi, Taihoku 1923, (229) 241-301). English Summary 1-18.

14. SMITH, M., and BEAVER, P., Persistence and distribution of **Toxocara** larvae in the tissues of children and mice. Pediatrics, 1953, 12, 491-496.

15. BEAVER, P., Parasitological Reviews. Larva migrans. Exptl. Parasitol, 1956, V, 587-621.

16. NICHOLS, R., The etiology of visceral larva migrans. I. Diagnostic morphology of infective second-stage **Toxocara** larvae. J. Parasitol., 1956, 42, 349-362.

17. NICHOLS, R., The etiology of visceral larva migrans. II. Comparative morphology of **Ascaris lumbricoides, Necator americanus, Strongyloides stercoralis** and **Ancylostoma caninum.** J. Parasitol., 1956, 42, 363-399.

18. SHOPE, R., The swine lungworm as a reservoir and intermediate host for swine influenza virus. I. The presence of swine influenza virus in healthy and susceptible pigs. II. The transmission of swine influenza virus by the swine lungworm. J. Exper. Med., 1941, 74, 41-68.

19. SYVERTON, J., McCOY, O., and KOOMEN, J., The transmission of the virus of lymphocytic choriomeningitis by **Trichinella spiralis.** J. Exper. Med., 1947, 85, 759-769.

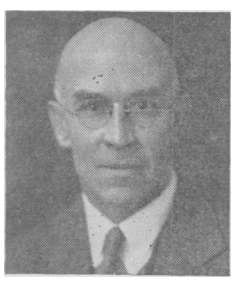
20. BEAUTYMAN, W., and WOOLF, A., An ascaris larva in the brain in association with acute anterior poliomyelitis. J. Pathol. Bacteriol. 1951, 63, 635-647.

OBITUARY

Dr. Edward A. Bruce

One of the best known veterinarians of the Pacific coast, indeed of all Canada, died on April 27th, 1958.

Dr. Bruce was born in Basutoland, Africa where his father was serving in the Royals Engineers. He was taken to Great Britain when a child and here his early education was obtained. He decided to follow the sea and took training, which led to an appointment as Midshipman. He served in this capacity for two years, finally coming to Canada in 1903. Soon thereafter he entered the Ontario Veterinary College, Toronto and after graduation established a practice in Newfoundland.



When the Dominion Government decided to establish meat inspection in Canada a group of young men were sent to Chicago for training. Bruce was one of these. Following the completion of this course he was appointed Travelling Inspector for the Meat Inspection Division and served in Ontario and the Eastern provinces. Tiring of this wandering life he requested an appointment giving a more fixed abode. Accordingly he was assigned to Calgary and later to Vancouver. In 1916 he was transferred to the Pathological Division and served under the late Seymour Hadwen. On Hadwen's appointment to the position of Chief Pathologist, Ottawa about 1918, Bruce was placed in charge of the branch laboratory and served in this capacity until his retirement in 1949. Following this he carried on pathological work for the Province on a part time basis.

There is left to mourn, his wife and two daughters of British Columbia and a brother and sister in England.

Dr. Bruce occupied a leading place in veterinary medicine in British Columbia. His early years in pathology were devoted to research problems. He has many publications to his credit. However it was in the field of service that Dr. Bruce made his greatest contribution. His knowledge of conditions in British Columbia was phenomenal. Because of this he was able to give helpful advice and guidance which were of great economic value.

In conclusion his many colleagues who found him a boon companion mourn his passing and express deep sympathy to his wife and family.